Chapter 27

Neuropsychological Assessment of Posttraumatic Stress Disorder (PTSD)

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Posttraumatic stress disorder (PTSD) is a mental disorder that sometimes develops after exposure to a life-threatening, psychologically traumatic event. Reflecting empirical advances relevant to the neurobiology and cognitive neuroscience of PTSD, this chapter will focus on PTSD as a neurobehavioral syndrome. We begin by describing PTSD, including a brief review of its clinical presentation and underlying neuropathology. We next review the neurocognitive characteristics of the disorder, common neuropsychological approaches to its assessment, and key clinical considerations in conducting neuropsychological evaluations when PTSD is a possible diagnosis. The chapter additionally addresses treatment implications, concluding with family and social considerations.

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Description of the Disorder

Diagnostic Criteria and Prevalence

Although numerous psychosocial and biological factors increase the risk of developing PTSD following exposure to a psychologically traumatic event [1–5], PTSD is unique among psychiatric disorders in that the diagnosis cannot be made without exposure to an environmental event (i.e., the trauma event). The fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) [6] defines a traumatic event as one in which a person "experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others" (Criterion A1) and had a subjective response that involved "intense fear, helplessness, or horror" in adults or "disorganized or agitated behavior" in children (Criterion A2). Epidemiological studies indicate that at least one of every two Americans (ages 15–54) have been exposed over the course of their lifetimes to a psychologically traumatic event and the majority of those exposed have faced two or more traumas in their lifetime [7].

As defined by DSM-IV, symptoms are grouped into three symptom criteria: (1) reexperiencing of the traumatic event (e.g., nightmares, physiological and emotional responsivity to trauma reminders); (2) avoidance of external reminders or thoughts

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associated with the trauma and numbing of general responsiveness (e.g., inability to have loving feelings); and (3) hyperarousal (e.g., concentration impairment, hypervigilance to threat, difficulty in sleeping). Factor analytic studies, however, suggest that a four-factor solution in which avoidance symptoms are separated from numbing and other symptoms may be more appropriate [8, 9]. DSM-IV additionally requires that symptoms endure for at least 1 month and cause clinically significant distress or functional impairment. Despite the frequency of trauma exposure, only about 25% of individuals confronted with trauma develop core PTSD symptoms [10]. The lifetime prevalence of PTSD among US adults has been estimated to be 6.8% [7]. Not surprisingly, prevalence is higher in at-risk populations such as combat veterans [11–14], inner-city children [15], and mass violence survivors [16].

Course and Associated Clinical Features

Course. PTSD typically begins with symptom emergence immediately following the traumatic event [17, 18], although it is possible for symptoms to have a delayed onset. Whereas a subset of individuals recover within a few months [19], PTSD can persist for decades or even for an individual's lifetime [20]. For example, approximately 90% of National Comorbidity Study (NCS) participants retrospectively reported that their PTSD symptoms were still present at 3 months, more than 70% continued to experience symptoms 1 year following the traumatic event, and more than one-third of the sample continued to experience PTSD symptoms 10 years or more, including those individuals who had received treatment [7]. Symptoms may also be cyclical, waxing and waning over time.

Comorbidities. PTSD rarely occurs in isolation from other emotional and behavioral symptoms. Kessler et al. [7], for example, reported that 88% of men and 79% of women with a lifetime diagnosis of PTSD met criteria for at least one other psychiatric diagnosis. Most common among these comorbid disorders are alcohol and substance use, mood, and non-PTSD anxiety disorders [21] (see Brady et al. for a review). Comorbidity rates of PTSD with other anxiety disorders (e.g., generalized anxiety disorder, panic disorder, simple phobia) ranged in the NCS from 7.3 to 31.4%, and lifetime prevalence rates of alcohol and drug use disorders were 51.9 and 34.5%, respectively, for men and 27.9 and 26.9%, respectively, for women with histories of PTSD [7]. Rates of comorbid major depression are likewise high, typically ranging from 30 to 50% (see [18]), with rates as high as 77% in treatment-seeking populations [22].

Traumatic stress exposures and PTSD also have been linked to health problems, such as cardio-and cerebrovascular disease, depressed immune functioning, pain disorders, increased health complaints, and decrements in health-related functioning [23–25]. Subsets of individuals diagnosed with PTSD may also experience physiological sleep abnormalities [26–29], potentially further damaging somatic, emotional, and cognitive health. Although some health problems may result from health risk behaviors such as increased tobacco use [28], as described below, others may be a direct consequence of neurobiological alterations.

Neurobiological Basis of PTSD

When confronted with life threat, the body responds with a state of physiological arousal, including acute increases in stress-related neurotransmitters and neuropeptides, such as corticotropin-releasing factor, norepinephrine, serotonin, dopamine, endogenous benzodiazepines, and endogenous opiates [30]. Although this response often serves an adaptive function in the immediate context of danger by facilitating actions that promote survival (i.e., "flight or fight" responses), the chronic dysregulation of these systems is believed to play an important role in both the pathogenesis and the maintenance of PTSD [31, 32] and is distinct from the pattern of neurobiological abnormalities associated with other stress-related psychiatric disorders such as anxiety and depression [32]. Unlike the profile of attenuated responsivity associated with habituation and adaptation to chronic stress [33] and major depression [34], PTSD is associated with

exaggerated neurobiological responsiveness to cues (now often harmless) of the original trauma [30] and the general sensitization of several neurobiological systems [32, 35]. This sensitization in turn can lead to over-responsiveness to subsequent stress and fear cues. Over time, the cumulative biological strain produced by repeated stress responses, known as "allostatic load," [36] can accelerate pathophysiology, including neuroimmune suppression and possibly neuronal damage.

Of particular relevance to the neuropsychology of PTSD is the dysregulation of the noradrenergic system, hypothalamic–pituitary–adrenal (HPA) axis, and serotonergic system [35]. These systems are believed to influence brain functioning in regions involved in the fear response, including the prefrontal cortex (PFC), amygdala, hippocampus, dorsal raphe nucleus, and locus coeruleus. In short, the combined dysregulation of these systems is thought to result in dampened prefrontal and hippocampal functioning and reduced medial prefrontal inhibition of the amygdala, a limbic structure central to fear-based emotion. Multiple reviews of the vast neurobiological literature relevant to PTSD are available [30, 35, 37–40].

Neuroimaging Findings

In this section, we present a brief overview of findings from structural and functional neuroimaging studies relevant to three critical brain regions (amygdala, medial prefrontal cortex, hippocampus) thought to be involved in the pathophysiology of PTSD. Several extensive reviews of these literatures are available [41–45].

Structural Imaging and Magnetic Resonance Spectroscopy (MRS)

Volumetric studies generally have revealed smaller hippocampal volumes in participants diagnosed with PTSD as compared to both no-PTSD trauma-exposed [43, 46–49] and non-trauma-exposed [43, 46–48, 50, 51] participants, although this finding has not been uniform [52], especially when samples with more recent trauma exposure were examined [53–55]. Gilbertson et al. [56] suggested that hippocampal volume may be a vulnerability for PTSD, rather than a consequence of the disorder, based on the finding that both the trauma-exposed and the non-trauma-exposed "co-twins" of veterans with PTSD showed smaller hippocampal volumes than non-PTSD trauma-exposed veterans and their non-exposed co-twins. Moreover, hippocampal volumetric differences may not become apparent until adulthood (see [43] for a review) and are not necessarily associated with neurocognitive performances, including on tasks of learning and memory [57]. Paralleling the volumetric findings, MRS studies examining the relative concentration of select compounds within the hippocampus have suggested that PTSD is associated with decreased neuronal health in the hippocampus [52, 58, 59].

A growing number of structural imaging studies have begun to examine the PFC and amygdala in relation to PTSD. In a meta-analysis of structural brain abnormalities in PTSD, Karl and colleagues [43] found significantly smaller left amygdala volumes in adults with PTSD compared to both healthy and trauma-exposed controls and significantly smaller anterior cingulate cortex compared to trauma-exposed controls. Not all studies examining amygdala volumes, however, have revealed differences between PTSD-diagnosed trauma survivors and comparison samples [47, 51, 53–55]. In contrast, those measuring frontal cortex volumes have revealed reduced volumes of the frontal cortex in PTSD [54, 60, 61], including decreased volumes in medial PFC structures [62–65] and reduced cortical thickness in much of the frontal gyri [66].

Functional Imaging

Functional neuroimaging studies of PTSD typically have demonstrated that individuals with PTSD, relative to comparison samples, show heightened amygdala responsivity and

deactivation or decreased activation of the hippocampal, anterior cingulate, and orbital frontal cortex in response to symptom provocation such as that elicited by combat sounds and trauma-related words [67–69], script-driven imagery [70–74], and administration of yohimbine, an alpha-2 adrenergic receptor antagonist [75]. A similar pattern has surfaced in studies using cognitive activation paradigms such as encoding and retrieval of threat-related words [76], the emotional Stroop task [77], and presentation and memory of emotional facial expressions [78–80]. Relationships between activation in the amygdala and medial prefrontal cortex in response to traumatic imagery [74, 81] and fear-related stimuli [80] suggest that these two structures are functionally related in PTSD.

Summary and Related Literatures

Although beyond the scope of this chapter, there is also significant evidence of electrophysiological abnormalities in PTSD suggestive of neural processing abnormalities to both neutral and trauma-relevant stimuli [82]. Taken together, multiple methodologies provide converging evidence of biological, physiological, and neuroanatomical abnormalities associated with PTSD that would be expected to be associated with neuropsychological impairment.

Neuropsychological Functioning in PTSD

Empirical Findings

In this section, we review the now sizable literature on clinical neuropsychological test performances in PTSD and organize our review by domains commonly assessed in clinical neuropsychological evaluations, emphasizing those domains with the strongest empirical bases. Other recent reviews are also available [83–87].

Intellectual functioning. PTSD in both children [88] and adults [89–96] is associated with lower estimated and omnibus IQ scores as compared to no-PTSD trauma-exposed and non-exposed comparison groups. Likewise, correlational studies indicate an inverse relationship between PTSD and intellectual performance, even after controlling for stressor severity [90, 94, 97]. Few studies have examined intellectual functioning comprehensively with multi-faceted tasks, but those that have suggested that performance on verbal, as compared to visual–spatial, intellectual tasks may be more strongly associated with PTSD status [88, 90, 95]. In children, intellectual performance decrements have been associated with both early trauma exposure [98] and cortisol-induced neuronal loss associated with trauma exposure [55].

Earlier work using archival records suggests a directional relationship in which higher IQ serves a protective role following trauma exposure, reducing risk of PTSD [94]. Gilberston et al. [99] provided additional support for this hypothesis by examining twin pairs composed of one Vietnam War-exposed and one non-exposed brother. Intellectual performance did not differ between trauma-exposed brothers and their non-exposed co-twins. Instead, no-PTSD exposed brothers and their non-exposed co-twins performed more proficiently on intellectual tasks than both exposed brothers with PTSD and their non-exposed co-twins. A recent study of combat veterans using archival data, however, suggests that this relationship may be more complex: pre-exposure intellectual performance appeared to be protective against development of PTSD symptoms only at lower levels of trauma severity [100]. Using combat exposure as an index of combat severity, at lower levels of combat exposure, pre-exposure intellectual scores were negatively correlated with post-exposure PTSD symptom levels. In contrast, at higher levels of trauma exposure, pre-combat intellectual performances were not significantly associated with post-exposure PTSD severity.

New learning and memory. Anterograde memory on episodic, declarative memory tasks is perhaps the most thoroughly examined

neuropsychological domain in the PTSD literature. Although several studies have yielded negative findings regarding the relationship of PTSD to anterograde memory functioning [101–103], the majority of studies have found that both children [104, 105] and adults [89–91, 93, 96, 106–117] with PTSD perform less proficiently than those without PTSD on one or more measures of learning or memory, with initial acquisition being the most frequently impaired aspect of memory dysfunction. There is also evidence of heightened sensitivity to proactive [118] and retroactive [96, 115, 117] interference in persons with PTSD. Whether PTSD is associated with degraded retention of newly learned information over longer delayed intervals is more ambiguous. Whereas PTSD was associated with less proficient memory retention in select studies [107], several studies failed to reveal PTSD-related deficits in memory retention [89, 91, 115, 119–121].

Two recent independent meta-analyses have attempted to address inconsistencies across studies through the advantages gained by pooling data. Both found that PTSD was associated with less proficient performance on verbal memory tasks. Brewin et al. [122] found small to moderate effect sizes for PTSD diagnostic status across different civilian and military trauma samples. The association between PTSD and memory impairment, which were more pronounced on verbal as compared to non-verbal memory tasks, could not be attributed to head injury and did not differ significantly according to immediate versus delayed recall conditions. Johnsen and Asbjornsen [111] extended these findings in a meta-analysis of immediate verbal memory performance, likewise finding a moderate effect size for PTSD diagnostic status. The effect was larger in military as compared to interpersonal trauma samples and when specific memory instruments (Wechsler Memory Scale subtests and the Rey Auditory Verbal Learning Test, as compared to the California Verbal Learning Test) were used.

Autobiographical memory. In addition to anterograde memory deficits, PTSD is associated with autobiographical memory abnormalities. On autobiographical memory tasks that require recall of a specific memory in response to a cue word, trauma survivors with PTSD, as compared to trauma-exposed participants without PTSD, are more likely to produce "over-general" memories (i.e., reflecting categories of events rather than a specific event) [123–126]. Overgeneral memory recall appears to be particularly pronounced for emotionally positive memories [124, 125, 127], suggesting a possible emotion-based cognitive bias. Although beyond the scope of this chapter, considerable controversy exists regarding whether traumatic autobiographical memories are encoded differently than non-traumatic memories or whether they differ only in the severity of impairment [128–132].

Attentional, executive, and prefrontal functioning. Despite the inclusion of concentration difficulties as a core PTSD diagnostic feature, PTSD does not appear to be associated with a general concentration deficit but instead appears to be associated with a specific pattern of attentional deficits. PTSD-related performance decrements have been documented repeatedly on working memory and divided attention tasks [89, 91, 95, 133, 134] and to a lesser extent [135] on tasks of sustained attention [96, 115, 136–138]. In contrast, some aspects of attention, such as shift of set (as measured by card sorting and visual selective attention tasks) and focus of attention (as measured by letter cancellation and the standard Stroop) appear to be relatively impervious to PTSD in non-elderly adults [93, 96, 115, 116, 136, 138–141], although PTSD-related deficits on card sorting tasks have been documented in elderly former prisoners of war [142] and children [104].

Contemporary neuroanatomical conceptualizations of PTSD implicate dysfunction of the prefrontal cortex, especially regarding its inhibitory functions. Consistent with this notion, PTSD has been shown to be associated with cognitive disinhibition [115, 140] and perseveration [99, 143]. Also suggesting prefrontal dysfunction, Vasterling et al. [116] found that, as compared to combat-exposed veterans without PTSD and non-combat-exposed veterans, Vietnam veterans with PTSD displayed relative performance deficits in olfactory recognition, a task sensitive to orbitofrontal integrity [144].

Language, visual–spatial, and motor functioning. The few studies examining basic language, visual– spatial, and motor functions in PTSD have failed to reveal PTSD-related deficits [145, 146] with the exception of performances on those tasks with a strong executive component, such as complex figural copying [92, 139, 147, 148], word list generation [104, 108, 118], and motor sequencing [92, 93]. Error analysis of clinical visuo-constructive tasks [149] and performance patterns on experimental visuo-spatial tasks [150] have also revealed PTSD-related deficits in processing local, as compared to global, stimulus attributes and distal contextual elements.

Summary. The existing literature indicates subtle, yet specific, cognitive deficits on tasks with significant executive demands (e.g., strategic learning, working memory, and inhibition tasks). Consistent with neuroimaging [46, 152], electrophysiological [153], and behavioral [149, 151] data implicating a cerebral asymmetry favoring the non-dominant hemisphere, neuropsychological studies of PTSD point to a modality-specific deficit in processing verbally mediated information. Although much of the neuropsychological literature relevant to PTSD is derived from non-elderly adult samples, existing studies of children and older adults suggest that the observed neuropsychological deficits are relatively consistent across the lifespan, although age may interact with PTSD such that the performance of older individuals possibly reflects aspects of both PTSD and aging [154, 155]. With rare exception [94, 99, 100], few studies have attempted to examine causal direction between cognitive dysfunction and PTSD in humans, leaving it an area ripe for further exploration via prospective methodology.

Implications for Clinical Evaluation

In our experience, PTSD referrals for neuropsychological evaluation typically center on requests to rule out alternative etiologies (e.g., degenerative disease, traumatic brain injury, cerebrovascular disease) for cognitive dysfunction and/or to document the extent of cognitive dysfunction associated with PTSD. Neuropsychological evaluation of PTSD patients can be used to inform treatment planning, including cognitive rehabilitation efforts. Occasionally, neuropsychologists also are referred cases in which the primary diagnosis of PTSD is not yet established or requires confirmation.

Confirming or establishing a PTSD diagnosis. As summarized in previous sections, empirical findings reveal that PTSD is associated with a pattern of mild cognitive deficits that are not necessarily specific to the disorder. Therefore, the primary diagnosis of PTSD is not made on the basis of neurocognitive testing, but instead requires the use of psychological assessment methods developed specifically for PTSD diagnosis. At its most basic level, the PTSD evaluation includes solicitation of the trauma event(s), assessment of the full range of PTSD symptoms and their linkage to the trauma event(s), and documentation of the duration and functional impact of the symptoms. State-of-the-art assessments typically incorporate multiple methods, including interview-based and paper-and-pencil self-report measures, allowing the examiner to capitalize on the strengths of each, while mitigating the relative weaknesses of each. An excellent summary of these measures can be found on the Department of Veterans Affairs National Center for PTSD website (www.ncptsd.va.gov).

Commonly employed structured interviews include measures focused solely on PTSD such as the Clinician-Administered PTSD Scale (CAPS) [156], Structured Interview for PTSD [157], the PTSD Symptom Scale Interview [158], and the PTSD Module of the Structured Clinical Interview for DSM-IV [159]. The CAPS is often considered the "gold standard" due to its inclusion of trauma assessment, linkage of symptoms to trauma events, assessment of associated features, and assessment of functional impact. Self-report measures often focus on symptom assessment and include those that are DSM-congruent, such as the PTSD Checklist [160], Davidson Trauma Scale [161], Impact of Events Scale – Revised [162], and the Posttraumatic Diagnostic Scale [163], as well as those that are considered less face valid because they do not show one-to-one DSM

symptom correspondence but as a result may be less specific to PTSD [164]. Examples of the latter group include the Mississippi Scale for Combat-Related Posttraumatic Stress Disorder [165], Los Angeles Symptom Checklist [166], Penn Inventory for Posttraumatic Stress Disorder [167], and the Trauma Symptom Inventory [168].

A smaller subset of measures with demonstrated psychometric properties have been designed for use with young trauma patients. Examples include the Clinician-Administered PTSD Scale for Children and Adolescents [169], Trauma Symptom Checklist for Children [170], Posttraumatic Stress Disorder Semi-Structured Interview and Observation Record [171], and the Child Post-Traumatic Stress Disorder Reaction Index [172]. Detailed discussions of the strengths and weaknesses of various adult and child assessment measures and approaches can be found elsewhere [173–176].

Assessment of comorbid conditions and other contributory factors. As described earlier, PTSD commonly is associated with other psychiatric and somatic disorders. Complicating the primary diagnosis, overlap in symptom criteria (e.g., concentration difficulties) between PTSD and other psychiatric (e.g., depression) and somatic (e.g., post-concussion syndrome) disorders often create diagnostic ambiguities. Comorbid conditions also potentially impact cognitive performance both directly and indirectly (through other mediating factors). For example, when accompanied by certain comorbidities (e.g., depression), trauma survivors may be at greater risk for suicidal and other harmful behaviors [177], some of which (e.g., gunshots wounds to the head, drug overdoses resulting in coma) may result in lasting neuropsychological impairment. Further, pharmacological treatment of PTSD and associated conditions may result in iatrogenic effects that either enhance or impair cognitive functioning, depending on the specific agent [178–182]. Finally, certain comorbidities (e.g., alcohol use disorders, depression, traumatic brain injury, sleep disturbance) may influence neuropsychological performance directly [134, 183–186].

As such, clinical neuropsychological evaluation of PTSD requires assessment of comorbid conditions (e.g., depression), health risk behaviors (e.g., suicide attempts, excessive alcohol consumption), and contextual factors (e.g., concurrent pharmacological treatment, sleep) that potentially complicate interpretation of the assessment data. When such complicating factors occur, it becomes important to document the timeline of their onset relative to the onset and course of PTSD as well as any neuropsychological deficits. For example, knowing the chronology of substance abuse in relation to the onset of cognitive impairment and PTSD symptoms may help determine that cognitive decline began only after substance use increased. This information in turn can be used to project prognosis under a range of different circumstances (e.g., once substance use is discontinued). Preliminary evidence that neurodevelopmental disorders (e.g., attention deficit hyperactivity disorder) may be associated with increased risk of PTSD [91–93] also highlights the need to assess mental disorders that predate PTSD onset. Likewise, neurobehavioral disorders (e.g., dementia) with onset postdating trauma exposure may be associated with recurrence or exacerbation of PTSD symptoms [187].

As an example of a complex clinical constellation, we highlight traumatic brain injury (TBI) occurring in the context of PTSD. TBI may have considerable overlap with PTSD in regard to neuropsychological deficits [188–198], associated somatic symptoms [199, 200], and underlying neural abnormalities [201, 202]. Depending on the relative severity of the two disorders, the overlap between PTSD and TBI on these dimensions can make differentiation of the relative contributions of each to neuropsychological deficits challenging [203]. Perhaps of greater relevance to the patient's day to day functioning, however, is that TBI may exacerbate existing PTSD and depression symptoms in trauma survivors [204–206], complicating the clinical presentation. In such cases, it becomes essential to understand the recency of the TBI(s), the onset of PTSD relative to the TBI(s), the relative severity of each disorder, and the degree to which there may be other complicating factors (e.g., headaches) that influence current cognitive

status and the course and prognosis of neuropsychological deficits.

Neurobehavioral Instrument Selection

As with neuropsychological evaluation of most disorders, we recommend incorporating at least cursory assessment of a broad range of cognitive domains, evaluating domains anticipated to be sensitive to PTSD diagnosis as well as those not expected to be affected. This approach allows evaluation of both confirmatory and disconfirmatory evidence of the hypothesized etiology of neuropsychological dysfunction and facilitates detection of non-PTSD etiologies. Screening multiple domains additionally identifies potential cognitive strengths that can be utilized to help compensate for observed deficits. Because the empirical literature suggests that PTSD-related deficits are relatively subtle, we recommend more comprehensive assessment of domains thought to be impaired in PTSD (e.g., learning, memory, inhibitory functions) using tasks that are reasonably challenging. Unfortunately, research examining neuropsychological functioning in PTSD only rarely has included assessment of effort, but the clinical context necessitates evaluation of cognitive effort for interpretation of the results. In the following paragraphs, we integrate findings from the empirical literature in considering clinical test selection in the two domains most commonly found to differ according to PTSD diagnosis (i.e., learning/memory and attention/executive functioning).

Learning and memory. Although both visual–spatial and verbal–auditory learning and memory deficits have been found to be associated with PTSD diagnosis, deficits have been more commonly documented on verbal–auditory tasks, and effect sizes appear larger on verbal–auditory as compared to visual–spatial tasks [122], suggesting that learning and memory should be assessed in both modalities. As with any disorder, it is typically useful to include both single- and multiple-exposure tasks, and to include tasks that assess both initial registration and retention. Although PTSD-related deficits have been observed on delayed recall [106], they less commonly have been observed when retention is computed relative to initial acquisition [91, 115, 116, 119, 154], suggesting that computation of difference scores or retention ratios may be clinically informative. Similarly, empirical findings indicating that PTSD is associated with heightened sensitivity to proactive and retroactive interference [115, 117, 118] suggest that tasks incorporating interference trials may provide clinically useful information.

The memory deficits associated with PTSD have been conceptualized as stemming in part from difficulties related to strategic learning [132, 207], highlighting the potential utility of administering tasks that vary in their demands on self-initiated strategy. For example, it may be helpful to compare performance on tasks with unrelated stimuli (placing additional burden on strategic memory processes) to performance on tasks in which there is an underlying categorical structure (demanding less strategic processing). Finally, we recommend analyzing errors (e.g., perseverations, intrusions) in memory assessments, given the mounting evidence that executive components of memory encoding and retrieval may be central to the memory deficit observed in PTSD.

Attentional and executive functions. One of the most theoretically interesting neuropsychological findings in PTSD (i.e., decreased response inhibition) may also be among the most clinically significant. The failure to gate information and regulate emotions strikes at the heart of PTSD with direct implications for the development and maintenance of the disorder. Specifically, it may be that frontally mediated deficits in inhibitory regulation influence how patients with PTSD process, encode, and retrieve trauma events and related memories [132]. Similarly, regulatory deficits of the limbic system have far-reaching implications for how emotions are experienced and managed. Therefore, we recommend that evaluation of attention/executive functions in PTSD include at a minimum a thorough assessment of response inhibition. Because

the full extent of executive and attentional deficits associated with PTSD is not yet fully understood (especially in terms of their interactions with developmental stage), neuropsychological evaluation of PTSD ideally will include a broad range of attention and executive tasks.

Summary. Neuropsychological evaluation of the PTSD patient poses specific challenges. Whereas there are observable group-level deficits on neurobehavioral measures, they are likely to be mild and difficult to interpret at the individual level. In addition, a number of other potential contributory factors (e.g., other medical conditions, medications, comorbid substance abuse) may complicate the clinical picture. However, even subtle deficits may have a significant impact on daily functioning [148]. Moreover, as described below under the "Treatment Implications" section, such deficits may also have as yet undocumented effects on treatment response, suggesting that neuropsychological evaluation of PTSD offers information of potential value to the overall clinical management of the patient.

The Emotional Stroop Paradigm

As suggested by the previous section, the performance deficits on standardized clinical neuropsychological tests that accompany PTSD are typically mild and overlap to some extent with comorbid disorders. In contrast to standardized neuropsychological assessment instruments, experimental information processing, electrophysiological, and functional imaging studies have yielded results suggesting that some types of information processing abnormalities may be specific to PTSD, especially when trauma-relevant stimuli are employed. In this section, we highlight the emotional Stroop task, an experimental paradigm that has been particularly robust in detecting information processing biases to trauma-relevant stimuli in PTSD [208]. Although we anticipate that functional imaging and electrophysiological paradigms will continue to generate findings that will move the field closer toward understanding the neuropsychology of PTSD, we focus on the emotional Stroop task because of its extensive empirical history and, because it does not require specialized equipment, its potential feasibility and widespread accessibility. The functional imaging literature has been reviewed briefly in previous sections, and comprehensive reviews of the electrophysiological and cognitive information processing PTSD literatures are available elsewhere [82, 209, 210].

The emotional Stroop is a variant of the Stroop color-naming task [211], in which respondents are shown color-congruent (e.g., the word "red" printed in red ink) and color-incongruent (e.g., the word "red" printed in blue ink) words and asked to name as quickly as possible the color of ink in which the word is printed. In the classic Stroop, respondents are slower to name color-incongruent words than color-congruent words [211]. The emotional Stroop variation modifies the paradigm by varying the emotional valence and relevance of the words (e.g., "chair" as a neutral word, "combat" as a trauma-relevant word for combat veterans). Slower naming of any particular class of words is interpreted as an attentional bias (i.e., an attentional preference or "pull") to the particular semantic category.

Relative to non-trauma-exposed and trauma-exposed individuals without PTSD, individuals with PTSD are slower to color-name trauma-related words as compared to emotionally neutral words or emotional words that are unrelated to their trauma [208, 212–216]. This attentional bias is thought to occur when the mild threat inherent to the semantic content of trauma words interferes with normal functioning and diverts cognitive resources to the threat-related information [208]. Attentional bias to trauma words in PTSD has been documented across a range of trauma populations, including rape victims [212, 213], combat veterans [214, 215, 217], and motor vehicle accident survivors [218, 219].

Although attentional biases to threat words on the Stroop have been well replicated, there continue to be several factors that limit its application as a clinical task. First, whereas idiographic lists are not required to show an effect, the trauma

words nonetheless need to be generally related to the respondent's trauma experience to elicit an effect. Thus, prior to clinical use, different versions of the task must be developed to accommodate diverse trauma populations. Second, and likely related to the diversity of stimuli necessary across trauma populations, clinical normative data do not yet exist. Such normative data will be critical, as biases to emotionally relevant words are not absolute but are instead relative to normal controls. Finally, theoretical debate continues regarding the parameters in which the emotional Stroop effect is most likely to occur and the degree to which it reflects automatic (i.e., involuntary and without conscious awareness or effort) versus strategic (i.e., requiring cognitive effort) processing [208, 220–222].

In sum, the emotional Stroop and other information processing paradigms continue to generate findings that elucidate the cognitive processes that underlie the development and perpetuation of PTSD (e.g., through reinforcement of fear networks) and explain PTSD symptoms such as hypervigilance and decreased concentration. Moreover, because of the specificity of attentional bias to threat-relevant information, some of these paradigms also hold potential for future clinical application; however, the field awaits further development of these tasks prior to widespread clinical implementation.

Treatment Implications

There are a number of psychosocial and psychopharmacological interventions used to treat PTSD. Below, we discuss the neuropsychological relevance of some of the more common of these interventions.

Pharmacological Treatment

Several psychotropic medications have been employed in the treatment of PTSD, including selective serotonin reuptake inhibitors (SSRIs), other antidepressants (e.g., tricyclics, monoamine oxidase inhibitors), anti-psychotic medications, and antiepileptic medications [180, 223]. However, the only two pharmacological agents approved by the US Food and Drug Administration specifically for treatment of PTSD are the SSRIs sertraline, and paroxetine [224]. Likewise, the Department of Veterans Affairs/Department of Defense Guidelines (VA/DOD) [225], the American Psychiatric Association [226], and the International Society of Traumatic Stress Studies (ISTSS) [227] endorsed SSRIs as the initial choice for the pharmacological treatment of PTSD. SSRIs impact multiple neurotransmitter systems [e.g., serotonin, glutamate, and gamma-aminobutyric acid (GABA)] that are thought to potentially impact cognitive functioning by improving inhibition of distracting recollections [223]. Supporting this hypothesis, preliminary evidence from single group designs suggests that SSRIs used in the treatment of PTSD may enhance performance on anterograde memory tasks [228] and alter neural activation from pre- to post-treatment in frontal, limbic, and paralimbic regions, particularly among treatment responders [229].

Psychotherapy

Psychological treatment approaches for PTSD include but are not limited to exposure-based interventions, cognitive–behavioral therapy, psychodynamic therapy, supportive counseling, anxiety management, and eye movement desensitization and reprocessing (EMDR). Of the many treatment approaches available, exposure-based and cognitive–behavioral interventions have been identified as the most efficacious in the treatment of PTSD [230–232], with exposure-based therapy named as the treatment of choice by the Institutes of Medicine [233].

Cognitive–behavioral interventions target modification of negative or distorted thoughts attached to trauma experiences, with the goal of generating more realistic explanations and thoughts associated with the trauma and trauma experience. Such modifications could be reasoned to require both the inhibition of

maladaptive thoughts and sufficient cognitive flexibility to reappraise thoughts and memories. In addition, the degree to which trauma memories can be retrieved and modified may be important to treatment, especially when exposure is included in the intervention. Although not directly measuring neurocognitive functioning, recent neuroimaging studies have demonstrated that activation levels of the amygdala and anterior cingulate cortex [234, 235] and anterior cingulate volumes [236] helped predict treatment response for cognitive–behavioral and exposure therapies, raising the question of whether associated cognitive functions also may be useful in predicting treatment response to common psychological PTSD interventions. Supporting this notion, Wild and Gur [237] reported that more proficient verbal encoding and recall performances at pre-treatment were associated with better PTSD treatment outcomes.

EMDR is a multi-phase treatment incorporating trauma visualization, simultaneous lateral eye movements, and the coupling of positive cognitions with trauma visualization [238], with the latter repeated until the patient reports a high level of belief in the positive cognition [239]. Thus, like exposure-based interventions and some forms of CBT, EMDR incorporates an exposure component. Significant debate exists regarding the incremental benefits of EMDR over other exposure-based interventions, and the underlying mechanism of change (i.e., imagined exposure, ocular movement) is not well understood [239–241].

Preliminary work suggests that cognitive–behavioral and other psychological interventions may alter neural functioning. Sutherland and Bryant reported improved recollection of specific memories and reduced recollection of overgeneral, categorical memories following cognitive–behavioral treatment, as well as reduced bilateral amygdala and anterior cingulate activation [126]. Although not measuring neuropsychological outcomes, in a randomized trial, Lindauer et al. likewise demonstrated changes in neural activation in frontal and paralimbic regions following brief eclectic psychotherapy as compared to a wait-list control condition [242].

Family Considerations

Neuropsychological evaluations often consider how disorders impact the family. In PTSD, emotional, and possibly cognitive, dysfunction may lead to significant disruptions of social and family functioning. PTSD patients have the most difficulty in their closest relationships, such as those with a partner or significant other and children. In intimate relationships, trauma survivors with PTSD report lower levels of marital satisfaction [243, 244], poor cohesion and expressiveness [244, 245], high levels of conflict, which sometimes include physical aggression [246–248], and less intimacy and sexual satisfaction [246, 249, 250]. In turn, their partners report significant marital problems and often show somatic symptoms, anxiety, depression, and insomnia [244, 251, 252].

Regarding relationships with their children, trauma survivors suffering PTSD, particularly those who experience high levels of PTSD numbing and avoidance symptoms, have poorer parent–child relationships and less satisfaction with parenting [253, 254]. Children who have a parent with PTSD live in households with significantly higher conflict and lower cohesiveness [245, 252]. These children are also more likely to have behavioral problems [244] and are at a greater risk for mental disorders, including PTSD [255]. Some literature suggests that partners and children may exhibit secondary PTSD in which family members take on some symptoms of PTSD [256–259].

These negative family outcomes are relevant not only to the family member but also to the patient, especially in light of robust findings that social support is a key resource and determinant of mental health outcomes for those suffering from PTSD [260, 261]. Because social relationships may be affected adversely by PTSD symptoms, without intervention, individuals with

PTSD may find themselves in a downward spiral in which one of the most valuable resources (i.e., social support) is less likely to be available. This reduction of social resources leads to ineffective coping behaviors (e.g., avoidance and isolation), which further deplete available resources, thus continuing the cycle. Therefore, in clinical settings, it is critical to assess family and social resources available to patients with PTSD. Fortunately, a number of interventions are emerging that may be particularly promising to address social dysfunction within intimate partner and family relationships [262–264].

Conclusions

Neuropsychological research, along with converging evidence from neurobiological, neuroimaging, and electrophysiological studies, suggests that the neural underpinnings of PTSD are integral to the disorder. Neuropsychological abnormalities include impairment of executive aspects of attention, sustained attention, learning, and memory. Performance on verbally mediated tasks, including IQ and anterograde memory tasks, is less likely to be proficient among trauma survivors who develop PTSD as compared to those who do not. The pattern of results is consistent with neuroanatomical models of PTSD that emphasize the prefrontal cortex and limbic/paralimbic areas, including the amygdala and the hippocampus. There is much about PTSD as a neurobehavioral disorder, however, that remains unresolved. For example, the degree to which neurobiological and neuropsychological abnormalities represent predispositional factors versus sequelae of trauma exposure is uncertain. Likewise, the extent to which comorbidity and treatment-related factors contribute to neuropsychological dysfunction in PTSD is not fully resolved. Inconsistencies in measurement and sampling methodology across studies have not permitted sufficient replication to create a highly delineated neuropsychological profile, although recent meta-analytic and longitudinal studies have begun to help address some of these issues. Finally, the addition of clinical neuropsychological measures within clinical trial research represents a particularly exciting application of neuropsychology. Inclusion of such assessment tools, both as outcome measures and as potential predictors of treatment response, will potentially have significant impact on the care of patients with PTSD.

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